centrations of benzene in the inhalations and exhalations (Fig 3), some 55–60% of the inhaled benzene being absorbed. A steady state of metabolism to phenol did not occur in two hours, and required in this subject 2½–3 hours (Figs 4, 5). The rates of excretion of urinary phenol expressed as mg/man/day for a continuing exposure to benzene vapour at a concentration of 100 mg/m³ approximate to 100 mg/man/day, a value of the order of that calculated for industrial exposures of this intensity (Docter & Zielhuis 1967).

In detecting exposures to the solvent benzene and estimating their intensity, the necessity for determinations made on man is obvious. The presence of benzene in expired air after exposure has ceased is definite evidence of exposure, but as yet the present studies are not able to relate the results of post-exposure analyses of expired air at known times after work with the concentrations of benzene respired at the workplace. More deliberate exposures to defined concentrations are needed. However, in determining the rates of excretion of urinary phenol for defined exposures to benzene vapour, exposures of four hours or more are essential, owing to the time required to reach a steady state of metabolism.

The early detection of absorption of toxic materials like solvents can be made by the analyses of tissues and body fluids for the presence of the materials or their metabolites. Where the toxic materials are absorbed in vapour form and excreted in vapour form, the analyses of post-exposure samples of expired air involve no difficult procedures for the workmen to undergo, give proof of exposures and may help in defining

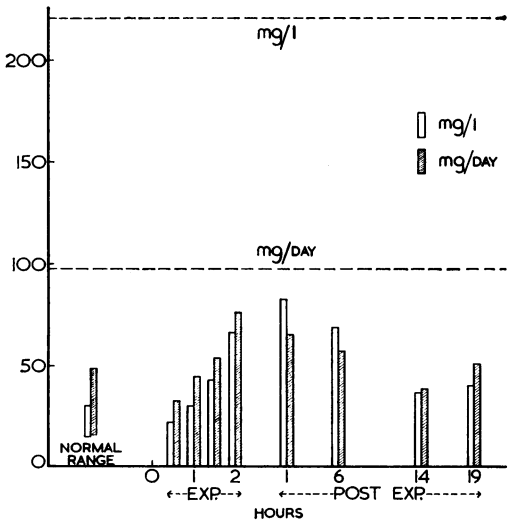

Fig 4 *Half-hourly urinary excretion of phenol during and after defined exposure to benzene vapour at a concentration of approximately 100 mg/m³ for 2 hours. Broken lines indicate calculated values after Docter & Zielhuis (1967)*

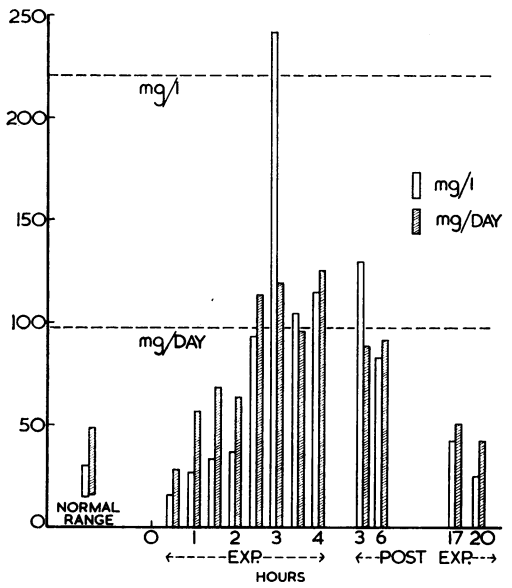

Fig 5 *Half-hourly urinary excretion of phenol during and after defined exposure to benzene vapour at a concentration of approximately 100 mg/m³ for 4 hours. Broken lines indicate calculated values after Docter & Zielhuis (1967)*

intensities. More research is needed to determine the pharmacodynamics of compounds of the nature of the toxic solvents in man, and in such studies the absorption and elimination of the parent compound and the elimination of its metabolites will be defined. For it is better to detect absorption in this fashion than to wait for pathological changes to announce industrial over-exposures.

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Comparative Merits of the Tests of Lead Exposure

Possible reasons for the divergence of opinion concerning the merits of various tests of lead exposure are tradition, expense, acceptability and lack of evidence. The ideal test should have a high correlation with lead exposure, or lead poisoning, whichever is being determined; a low variability, because if the scatter is small more weight can be given to an estimation; and, for screening, a low cost because more readings can be obtained on a given budget.

The comparative merit of each test for estimating lead exposure may be calculated as the slope

of the regression line on lead-in-air concentration, divided by the standard deviation about the line. A survey has been made in an electric accumulator factory (Williams *et al.* 1968). Thirty-nine lead workers and controls whose lead absorption was fairly stable wore personal samplers every working day for two weeks to obtain estimates of their exposure. During the second week six biological tests were each estimated daily. Blood lead was found to have the greatest merit followed by urinary lead and coproporphyrin together. Urinary δ-aminolævulinic acid was of less merit, the punctate basophil count of little merit and hæmoglobin of no merit at the level of exposure studied. Urinary coproporphyrin had the greatest merit per unit cost.

Perhaps the method has other applications.

REFERENCE
Williams M K, King E & Walford J (1968) *Brit. med. J.* i, 618

Dr H Loewenthal (*London*) said that no laboratory test on its own could distinguish between excessive and early toxic absorption of lead. Even very high blood lead levels did not necessarily indicate lead poisoning, but hæmatological and biochemical tests taken together would facilitate diagnosis by the factory doctor. The wider use of the polarographic method of lead estimation and the use of the micro-method for the packed cell volume estimation would require smaller quantities of blood and this would help to allay the apprehension of the lead worker who did not object so much to the withdrawal of blood from his vein as to the amount which had to be taken if standard methods were used in the laboratory.

Dr Owen McGirr (*London Airport*) asked Dr Hunter if his Fig 1, demonstrating twenty-four-hour excretion of phenols after a relatively short inhalation of benzene, could be considered as an expression of a logarithmic relationship indicating a time-dose relationship. If so, this was perhaps comparable to biological responses to some physical exposures such as ionizing radiations and intense noise. He noted that even at twenty-four hours there remained a (small) body burden of phenols and wished to know whether successive exposures would increase the phenolic body burden.

Dr Hunter, in reply, said that after a single inhalational exposure to benzene vapour only one-third of the absorbed dose was eliminated in the form of urinary phenol. The urinary phenol fell to within the normal range within 24–36 hours. Therefore the residual burden of benzene must be metabolized very slowly and the body must retain a burden of benzene for each exposure rather than a burden of phenol. The benzene dose/phenol excretion time relationships were not known and relationships in Fig 1 were those for one subject.

Dr J R Glover (*Institute of Preventive Medicine, Cardiff*) said there appeared to be some confusion between absorption of toxic substances and poisoning by those toxic substances. The position was shown diagrammatically in Fig 1.

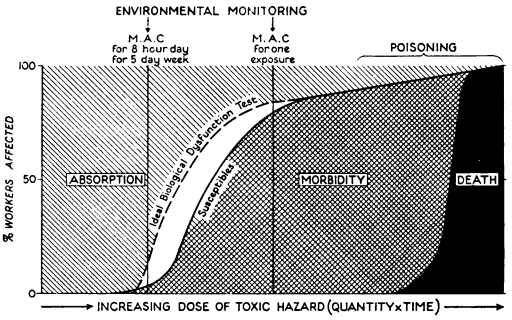

Fig 1 *A model to illustrate the relationship between absorption and poisoning*

Almost every hazard had a group of the population who were particularly susceptible to it and a group who were particularly resistant to it. The 'susceptible' graph was therefore S-shaped. For example, the maximum allowable concentration (MAC) of selenium was 0·1 mg Se/m³, However, a person susceptible to skin contact with selenium dioxide would show skin signs in atmospheres where selenium was so low that it could not be measured. 'Morbidity' on Fig 1 might be defined as signs and symptoms caused by the toxic hazard. This type of diagram was useful for deciding whether a test of biological dysfunction was too sensitive or too late in picking up early cases of poisoning by the toxic hazard.

Meeting June 23 1967

The meeting took the form of a visit to the Victoria Line, London Transport, which is under construction, and included Oxford Circus Station, Cobourg Street switch house, substation and control room, and Euston Station.

Meeting October 26 1967

Professor W Melville Arnott (*Queen Elizabeth Hospital, Birmingham*) delivered his Presidential Address which was entitled **Caring for the Worker.**

Meeting December 14 1967

A discussion was held on the subject of **The Early Recognition of Environmental Hazards.** The opening speakers were Dr A M Adelstein, Dr H H Pilling and Dr J C Gilson.